



The NFL-TBS.40-63 peptide targets and kills glioblastoma stem cells derived from human patients and also targets nanocapsules into these cells

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Titre	The NFL-TBS.40-63 peptide targets and kills glioblastoma stem cells derived from human patients and also targets nanocapsules into these cells
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Auteur	Lépinoux-Chambaud, Claire [1], Eyer, Joël [2]
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Mots-clés	Glioblastoma [3], Peptide [4], Prolifération [5], Stem Cells [6], targeting [7]
Résumé en anglais	<p>Glioblastoma stem cells correspond to brain tumor-initiating cells (BTICs) that have been identified in glioblastoma, the most common and aggressive brain tumor, as responsible for tumor initiation, progression and recurrence due to their resistance to current treatments. Targeting these cancer stem cells represents a crucial challenge to develop new therapeutic strategies. Previous works have shown that the NFL-TBS.40-63 peptide, corresponding to a tubulin-binding site on neurofilaments, targets and reduces in vitro and in vivo the viability of glioblastoma cells without affecting healthy cells. The objective of this study is to investigate the effect of this peptide on BTICs isolated from human glioblastoma. The uptake of this peptide alone or coupled to nanocapsules was analyzed by flow cytometry and immunochemistry. Its anti-tumor effect was studied using proliferation, adhesion and viability assays. Peptide-mediated effects were also evaluated on the BTIC self-renewal ability and by immunocytochemistry to investigate their cell shape and microtubule network. Here we show that the peptide enters massively in BTICs and demonstrates an anti-tumor effect by inhibiting their proliferation and inducing their death through an alteration of their microtubule network and cell-cell adhesion, and a decrease in the self-renewal ability of these cancer stem cells. These results indicate that the NFL-TBS.40-63 peptide represents a promising therapeutic drug for glioblastoma treatment by targeting and killing both glioblastoma cells and BTICs to prevent recurrence.</p>
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Liens

- [1] <http://okina.univ-angers.fr/c.lepinoux/publications>
- [2] <http://okina.univ-angers.fr/joel.eyer/publications>
- [3] <http://okina.univ-angers.fr/publications?f%5Bkeyword%5D=8332>
- [4] <http://okina.univ-angers.fr/publications?f%5Bkeyword%5D=14658>
- [5] <http://okina.univ-angers.fr/publications?f%5Bkeyword%5D=14653>
- [6] <http://okina.univ-angers.fr/publications?f%5Bkeyword%5D=16879>
- [7] <http://okina.univ-angers.fr/publications?f%5Bkeyword%5D=19131>
- [8] <http://okina.univ-angers.fr/publications/ua19643>
- [9] <http://dx.doi.org/10.1016/j.ijpharm.2019.05.060>
- [10] <https://www.sciencedirect.com/science/article/pii/S0378517319304193?via%3Dihub>
- [11] <http://www.ncbi.nlm.nih.gov/pubmed/31132447?dopt=Abstract>

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